```
     (FILE 'HOME' ENTERED AT 09:58:35 ON 30 NOV 2004)

     FILE 'REGISTRY' ENTERED AT 10:02:29 ON 30 NOV 2004
L1             1 S QUERCETIN/CN
L2             1 S GENISTEIN/CN

     FILE 'USPATFULL, CAPLUS' ENTERED AT 10:08:16 ON 30 NOV 2004

     FILE 'USPATFULL, CAPLUS, EMBASE' ENTERED AT 10:08:22 ON 30 NOV 2004
L3          1649 FILE USPATFULL
L4         14625 FILE CAPLUS
L5          5730 FILE EMBASE
     TOTAL FOR ALL FILES
L6         22004 S QUERCETIN OR L1
L7           135 FILE USPATFULL
L8            45 FILE CAPLUS
L9            23 FILE EMBASE
     TOTAL FOR ALL FILES
L10           203 S L6 AND SCAR?
L11           147 FILE USPATFULL
L12            46 FILE CAPLUS
L13            23 FILE EMBASE
     TOTAL FOR ALL FILES
L14           216 S L6 AND (SCAR? OR CICATRI?)
L15            18 FILE USPATFULL
L16            24 FILE CAPLUS
L17            16 FILE EMBASE
     TOTAL FOR ALL FILES
L18            58 S L6 (1S) (SCAR? OR CICATRI?)
L19            31 DUP REM L16-L17 (9 DUPLICATES REMOVED)
L20          1459 FILE USPATFULL
L21          7647 FILE CAPLUS
L22          6243 FILE EMBASE
     TOTAL FOR ALL FILES
L23         15349 S GENISTEIN OR L2
L24             6 FILE USPATFULL
L25             6 FILE CAPLUS
L26            11 FILE EMBASE
     TOTAL FOR ALL FILES
L27            23 S L23 (1S) (SCAR? OR CICATRI?)
L28            14 DUP REM L25-L26 (3 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 10:24:58 ON 30 NOV 2004

=>
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L6 ANSWER 5 OF 25 CAPLUS COPYRIGHT 2005 ACS on STN
IT **Acne**
Cell differentiation
Sebum
Sunscreens
(skin care compns. containing naringenin and/or **quercetin** and retinoid)
AN 1997:609663 CAPLUS
DN 127:267821

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5665367	A	19970909	US 1996-722540	19960927

L6 ANSWER 6 OF 25 CAPLUS COPYRIGHT 2005 ACS on STN
AB The effects of protein kinase C (PKC) activator 1-O-tetradecanoyl phorbol-13-acetate (TPA) and inhibitor 1-(5-isoquinolinylsulfonyl)-2-methylpiperazine (H-7) and **quercetin** were studied on release of tumor necrosis factor (TNF) from mouse peritoneal macrophages primed with Propionibacterium **acnes** (PA). TPA (1-100 ng/mL) and lipopolysaccharides (LPS) (1-100 ng/mL) induced the release of TNF from PA-primed mouse peritoneal macrophages in. . . dose- and time-dependent manners in vitro, and the effects of PTA and LPS were inhibited by H-7 (12.5-100 μmol/L) or **quercetin** (6.25-25 μmol/L) in a dose-dependent manner. After i.p. H-7 (50 mg/kg), LPS-induced release of TNF in vivo decreased significantly. These. . .
AN 1993:189733 CAPLUS
DN 118:189733

L6 ANSWER 7 OF 25 CAPLUS COPYRIGHT 2005 ACS on STN
ST sebaceous gland inhibitor isoflavone; **acne** treatment isoflavone; **genistein acne** treatment
AN 1991:17588 CAPLUS
DN 114:17588

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 02193919	A2	19900731	JP 1989-13481	19890123

L6 ANSWER 8 OF 25 CAPLUS COPYRIGHT 2005 ACS on STN
AB Microbicidal formulations, especially effective against P. **acnes** on the skin, contain **quercetin** (I) [117-39-5] in combination with rutin (II) [153-18-4] and/or isorhamnetin 3-O-rutinoside (III) [604-80-8]. Thus, I 0.6, II 2.0, and III. . .
ST quercetin isorhamnetin rutin microbicide; Propionibacterium **quercetin** isorhamnetin rutin; **acne** flavonoid
IT Propionibacterium **acnes**
(control of, **quercetin** and isorhamnetin rutinoside and rutin for)
IT **Acne**
(treatment of, with skin lotion containing isorhamnetin rutinoside and **quercetin** and rutin)
IT 153-18-4
RL: BIOL (Biological study)
(skin lotion containing isorhamnetin rutinoside and **quercetin** and, for **acne** treatment)
IT 604-80-8
RL: BIOL (Biological study)
(skin lotion containing **quercetin** and rutin and, for **acne** treatment)
AN 1984:460134 CAPLUS
DN 101:60134

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 59044313	A2	19840312	JP 1982-154598	19820907
JP 02001806	B4	19900112		

L6 ANSWER 9 OF 25 CAPLUS COPYRIGHT 2005 ACS on STN
AB . . . disks, and it was found that the EtOH extract of flower buds of S. japonica showed antibacterial activity against P. **acnes**, P. avidum, and Staphylococcus aureus under weak acidic conditions. After purification of the extract, the activity was attributed to the interaction caused by 3 components: **quercetin** (I), rutin (II), and isorhamnetin-3-rutinoside (III). When I, II, and III were applied singly, only I showed very weak activity,. . .
AN 1984:451606 CAPLUS
DN 101:51606

Experimental Cell Research
Volume 228, Issue 1 , 10 October 1996, Pages 29-35

doi:10.1006/excr.1996.0295 Cite or Link Using DOI

Regular Article

Integrin $\alpha 2\beta 1$-Dependent Contraction of Floating Collagen Gels and Induction of Collagenase Are Inhibited by Tyrosine Kinase Inhibitors

Arsi Broberg and Jyrki Heino [1]

MediCity Research Laboratory and Department of Medical Biochemistry, University of Turku, Tykistökatu 6A, FIN-20520, Turku, Finland

Received 29 January 1996; revised 13 June 1996. Available online 19 April 2002.

Abstract

A cell culture inside a three-dimensional gel of fibrillar collagen is an experimental model used to study the response of cells to the extracellular matrix. Many cell types induce the contraction of gel and simultaneously decrease their production of type I collagen, whereas the expression of interstitial collagenase (matrix metalloproteinase-1; MMP-1) is enhanced. We have previously shown that in osteogenic cells the collagen receptor $\alpha 2\beta 1$ integrin is a positive regulator of MMP-1 and that the number of $\alpha 2\beta 1$ integrins on the cell surface also regulates the magnitude of contraction. However, the downregulation of collagen mRNA levels is not initiated by $\alpha 2\beta 1$ integrin. Here, we have studied in human KHOS-240 and MG-63 osteosarcoma cells and in human skin fibroblasts the effects of tyrosine kinase inhibitors on collagen gel contraction and on the regulation of MMP-1 and collagen $\alpha 1$(I) genes by extracellular collagen. The induction of MMP-1 could be inhibited by all tyrosine kinase inhibitors tested with the exception of genistein. None of them could prevent the downregulation of collagen expression. Thus, the collagen-induced alterations in the expression of MMP-1 and collagen $\alpha 1$(I) seem to be dependent on distinct signal transduction pathways. Many of the inhibitors, including genistein, could prevent the contraction of collagen gels. The effect was not related to their ability to inhibit cell growth, because an inhibitor specific for DNA synthesis and cell division did not have the same

effect. Thus, we suggest that the process of collagen gel contraction requires protein-tyrosine phosphorylation and that the ability of cells to contract collagen gels is not related to the induction of MMP-1 or to the level of collagen α1(I) expression. Finally, we propose that the tyrosine kinase inhibitors might be considered as candidate molecules in the treatment of pathological scar contraction.

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Experimental Cell Research
Volume 228, Issue 1 , 10 October 1996, Pages 29-35

hyperdictionary

English Dictionary | Computer Dictionary | Thesaurus | Dream Dictionary | Medical Dictionary

Search Dictionary: scar Search

Meaning of SCAR

Pronunciation: skÃ¢r

WordNet Dictionary

Definition:

1. [n] an indication of damage
2. [n] a mark left (usually on the skin) by the healing of injured tissue
3. [v] mark with a scar; "The skin disease scarred his face permanently"

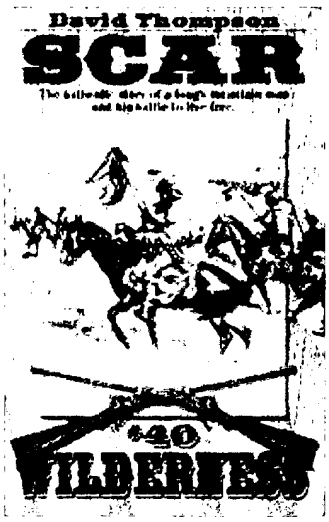
Websites:

Synonyms: cicatrice, cicatrix, mark, mark, pit, pock, scrape, scratch

See Also: blemish, blemish, callus, cheloid, cicatrise, cicatrize, deface, defect, disfigure, incise, keloid, pockmark, sw symptom, vaccination

Products Dictionary

Definition: **Scar**



Description not available.

more details ...

Webster's 1913 Dictionary

Definition:

1. \Scar\, n. [OF. escare, F. eschare an eschar, a dry slough (cf. It. & Sp. escara), L. eschara, fr. Gr. ? hearth, fireplace, scab, eschar. Cf. {Eschar}.]
   1. A mark in the skin or flesh of an animal, made by a wound or ulcer, and remaining after the wound or ulcer is healed; a cicatrix; a mark left by a previous injury; a blemish; a disfigurement.

      This earth had the beauty of youth, . . . and not a wrinkle, scar, or fracture on all its body. --T. Burnet.

   2. (Bot.) A mark left upon a stem or branch by the fall of a leaf, leaflet, or frond, or upon a seed by the separation of its support. See Illust.. under {Axillary}.

2. \Scar\, v. t. [imp. & p. p. {Scarred}; p. pr. & vb. n. {Scarring}.]
   To mark with a scar or scars.

   Yet I'll not shed her blood; Nor scar that whiter skin of hers than snow. --Shak.

   His cheeks were deeply scarred. --Macaulay.

3. \Scar\, v. i.
   To form a scar.

4. \Scar\, n. [Scot. scar, scaur, Icel. sker a skerry, an isolated rock in the sea; akin to Dan. ski[ae]r, Sw. sk["a]r. Cf. {Skerry}.]
   An isolated or protruding rock; a steep, rocky eminence; a bare place on the side of a mountain or steep bank of earth. [Written also {scaur}.]

   O sweet and far, from cliff and scar, The horns of Elfland faintly blowing. --Tennyson.

5. \Scar\, n. [L. scarus, a kind of fish, Gr. ska`ros.] (Zo["o]l.)
   A marine food fish, the scarus, or parrot fish.

Dream Dictionary

Definition: Seeing a scar in your dream, symbolizes struggles and/or painful memories and bad feelings which may h entirely healed and still continue to linger in your mind. It suggests that your past still has some influence your life. Alternatively, a scar may represent deep-seeded insecurities which may be holding you back fror your goals.

Biology Dictionary

Definition: A scar is an abnormal condition of the skin after injury. It is characterized by dense fibrotic tissue covered epidermis.

HOME | ABOUT H

L19 ANSWER 25 OF 31 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AB . . . is mitogenic for fibroblasts and a stimulatory factor for collagen synthesis. Objectives: We have assessed the in vitro effects of **quercetin** on proliferation, collagen synthesis and the expression of the IGF system in keloid-derived fibroblasts. Methods Fibroblasts were isolated from earlobe keloids and exposed to **quercetin** at different concentrations. The inhibitory effects of **quercetin** on fibroblast proliferation were assayed using 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay, Western and Northern blot analyses. Results: **Quercetin** inhibited keloid fibroblast (KF) proliferation in a dose-dependent manner. Significant growth inhibition was observed on day 2 of culture. The. . . growth inhibition was approximately 25 μg mL(-1). Collagen 1 expression was significantly decreased while collagen 3 was almost undetectable following **quercetin** treatment. Basal levels of IGF-I receptor (IGF-IR) β subunits, p85 subunit of phosphatidylinositol 3-kinase, c-Raf, phospho-Raf-1, phosphoMEK 1/2, phospho-mitogen-activated protein kinase, phospho-Elk-1 and phospho-Akt-1 were significantly reduced when KF cells were exposed to **quercetin** for 24 h. Blocking IGF-IR activity with IGF-IR antibody or neutralizing endogenous IGF-I activity with IGF-I antibody led to significant. . . Conclusions: Because the IGF system plays an important part in fibroblast cell proliferation and collagen production, the described activities of **quercetin** on the IGF system and collagen expression may provide a novel approach for the use of **quercetin** in treatment and/or prevention of hypertrophic **scar** and keloid.

AN 2003166605 EMBASE

TI Suppression of insulin-like growth factor signalling pathway and collagen expression in keloid-derived fibroblasts by quercetin: Its therapeutic potential use in the treatment and/or prevention of keloids.

AU Phan T.T.; See P.; Tran E.; Nguyen T.T.T.; Chan S.Y.; Lee S.T.; Huynh H.

CS H. Huynh, Lab. of Molecular Endocrinology, Div. of Cell. and Molecular Research, National Cancer Centre of Singapore, Singapore 169610, Singapore. cmrhth@nccs.com.sg

SO British Journal of Dermatology, (1 Mar 2003) 148/3 (544-552).
Refs: 49
ISSN: 0007-0963 CODEN: BJDEAZ

CY United Kingdom

DT Journal; Article

FS 013 Dermatology and Venereology
030 Pharmacology
037 Drug Literature Index

LA English

SL English

L19 ANSWER 26 OF 31 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AB Currently accepted conservative treatments of hypertrophic **scars** are limited to steroid injections, radiation therapy, and silicone occlusive therapy. However, the use of Mederma for these problematic lesions. . . quite prevalent in the clinical setting. Little scientific evidence exists to support the efficacy of this product in reducing hypertrophic **scars**. The aim of this study was to study the effects of Mederma on hypertrophic **scars** in the rabbit hypertrophic **scar** model, allowing the histologic quantification of **scar** elevation, dermal collagen organization, vascularity, and inflammation and the gross examination of **scar** erythema. Full-thickness wounds down to cartilage, four per ear, were created in four New Zealand White rabbits, for a total of 32 **scars**. Twenty-eight days after the initial wounding, the hypertrophic **scars** were photographed, and treatment of half of the

scars on each ear was begun with Mederma three times per day for a total of 4 weeks. The untreated **scars** served as control **scars** and were left exposed to air. After 4 weeks of treatment, the **scars** were once again photographed. The rabbits were then killed, and the **scars** were analyzed histologically. The pretreatment and posttreatment photographs were compared by using computer quantification of magenta, yellow, and cyan expression within the **scars**. Histologic analysis demonstrated no significant reduction in **scar** hypertrophy or **scar** elevation index. However, a significant improvement in dermal collagen organization was noted on comparing Mederma-treated **scars** with untreated control **scars** ($p < 0.05$). No significant difference in dermal vascularity or inflammation was noted. Computer analysis of the **scar** photographs demonstrated no significant reduction in **scar** erythema with Mederma treatment. The active product in Mederma, allium cepa, has as its derivative **quercetin**, a bioflavonoid noted for its antiproliferative effects on both normal and malignant cells, and its antihistamine release effects. These properties could theoretically prove beneficial in reversing the inflammatory and proliferative responses noted in hypertrophic **scars**. Despite the authors' inability to demonstrate a reduction in **scar** hypertrophy, the improvement in collagen organization noted in the Mederma-treated **scars** suggests it may have an effect on the pathophysiology of hypertrophic **scar** formation.

AN 2002229561 EMBASE
TI Effect of Mederma on hypertrophic scarring in the rabbit ear model.
AU Saulis A.S.; Mogford J.H.; Mustoe T.A.
CS Dr. T.A. Mustoe, Division of Plastic Surgery, Northwestern Memorial Hospital, Galter Pavilion, 675 North Saint Claire, Chicago, IL 60611, United States. tmustoe@nmh.org
SO Plastic and Reconstructive Surgery, (2002) 110/1 (177-183).
Refs: 20
ISSN: 0032-1052 CODEN: PRSUAS
CY United States
DT Journal; Article
FS 009 Surgery
013 Dermatology and Venereology
037 Drug Literature Index
LA English
SL English

L29 ANSWER 98 OF 191 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 6
AN 1999:135047 CAPLUS
DN 130:279811
ED Entered STN: 03 Mar 1999
TI Lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells
AU Dixon, Richard J.; Young, Ken; Brunskill, Nigel J.
CS Department of Cell Physiology and Pharmacology, Leicester University School of Medicine, Leicester, LE1 9HN, UK
SO American Journal of Physiology (1999), 276(2, Pt. 2), F191-F198
CODEN: AJPHAP; ISSN: 0002-9513
PB American Physiological Society
DT Journal
LA English
CC 13-6 (Mammalian Biochemistry)
AB Patients with proteinuria tend to develop progressive renal disease with proximal tubular cell atrophy and interstitial **scarring**. It has been suggested that the nephrotoxicity of albuminuric states may be due to the protein mol. itself or by lipids, such as lysophosphatidic acid (LPA), that albumin carries. LPA was found to cause a transient increase in intracytoplasmic free Ca2+ ([Ca2+]i) in opossum kidney proximal tubule cells (OK) that was maximal at 100 μM LPA and was dose dependent with an EC50 of 2.6+10-6 M. This Ca2+ mobilization was from both internal stores and across the plasma membrane and was pertussis toxin (PTX) insensitive. Treatment of OK cells with 100 μM LPA for 5 min was found to cause a twofold increase in [3H]thymidine incorporation and a three- to fivefold increase over control after 24 h. This was highly PTX sensitive and insensitive to pretreatment with the tyrosine kinase inhibitors **genistein** and herbimycin A. These findings may be of significance in the progression of renal disease and indicate the potential importance of lipids in modulating proximal tubule cell function and growth.
ST lysophosphatidate calcium proliferation kidney proximal tubule
IT Animal cell line
(OK; lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells)
IT Cell proliferation
Signal transduction, biological
(lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells)
IT Lysophosphatidic acids
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells)
IT G proteins (guanine nucleotide-binding proteins)
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(pertussis toxin-insensitive; lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells)
IT G proteins (guanine nucleotide-binding proteins)
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(pertussis toxin-sensitive; lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells)
IT Kidney
(proximal tubule; lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells)
IT Albumins, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)

(serum; lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells)

IT 80449-02-1, Protein tyrosine kinase
RL: BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells)

IT 9002-64-6, Parathormone 62229-50-9, EGF
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells)

IT 7440-70-2, Calcium, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(lysophosphatidic acid-induced calcium mobilization and proliferation in kidney proximal tubular cells)

IT 7440-70-2, Calcium, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

L29 ANSWER 99 OF 191 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 7
AN 1996:736530 CAPLUS
DN 126:29610
ED Entered STN: 14 Dec 1996
TI Integrin α2β1-dependent contraction of floating collagen gels and induction of collagenase are inhibited by tyrosine kinase inhibitors
AU Broberg, Arsi; Heino, Jyrki
CS MediCity Research Lab. Dep. Med. Biochemistry, Univ. Turku, Turku, FIN-20520, Finland
SO Experimental Cell Research (1996), 228(1), 29-35
CODEN: ECREAL; ISSN: 0014-4827
PB Academic
DT Journal
LA English
CC 13-6 (Mammalian Biochemistry)
AB A cell culture inside a three-dimensional gel of fibrillar collagen is an exptl. model used to study the response of cells to the extracellular matrix. Many cell types induce the contraction of gel and simultaneously decrease their production of type I collagen, whereas the expression of interstitial collagenase (matrix metalloproteinase-1; MMP-1) is enhanced. The authors have previously shown that in osteogenic cells the collagen receptor α2β1 integrin is a pos. regulator of MMP-1 and that the number of α2β1 integrins on the cell surface also regulates the magnitude of contraction. However, the downregulation of collagen mRNA levels is not initiated by α2β1 integrin. Here, the authors have studied in human KHOS-240 and MG-63 osteosarcoma cells and in human skin fibroblasts the effects of tyrosine kinase inhibitors on collagen gel contraction and on the regulation of MMP-1 and collagen α1(I) genes by extracellular collagen. The induction of MMP-1 could be inhibited by all tyrosine kinase inhibitors tested with the exception of **genistein**. None of them could prevent the downregulation of collagen expression. Thus, the collagen-induced alterations in the expression of MMP-1 and collagen α1(I) seem to be dependent on distinct signal transduction pathways. Many of the inhibitors, including **genistein**, could prevent the contraction of collagen gels. The effect was not related to their ability to inhibit cell growth, because an inhibitor specific for DNA synthesis and cell division did not have the same effect. Thus, the authors suggest that the process of collagen gel contraction requires protein-tyrosine phosphorylation and that the ability of cells to contract collagen gels is not related to the induction of MMP-1 or to the level of collagen α1(I) expression. Finally, the authors propose that the tyrosine kinase inhibitors might be considered as candidate mols. in the treatment of pathol. **scar** contraction.
ST collagen contraction collagenase tyrosine kinase integrin
IT Extracellular matrix
Second messenger system
(integrin α2β1-dependent contraction of floating collagen gels and induction of collagenase are inhibited by tyrosine kinase inhibitors)
IT Collagens, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(type I; integrin α2β1-dependent contraction of floating collagen gels and induction of collagenase are inhibited by tyrosine kinase inhibitors)
IT Integrins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(α2β1; integrin α2β1-dependent contraction of floating collagen gels and induction of collagenase are inhibited by tyrosine kinase inhibitors)
IT 80449-02-1, Kinase (phosphorylating), protein (tyrosine)
RL: BAC (Biological activity or effector, except adverse); BSU (Biological

study, unclassified); BIOL (Biological study)
(integrin α2β1-dependent contraction of floating collagen gels and induction of collagenase are inhibited by tyrosine kinase inhibitors)

IT 9001-12-1, Collagenase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(integrin α2β1-dependent contraction of floating collagen gels and induction of collagenase are inhibited by tyrosine kinase inhibitors)

RE.CNT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD

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L36 ANSWER 70 OF 149 USPATFULL on STN

SUMM In some of its method aspects, the present invention provides methods for **treating** cardiovascular disease, **treating** inflammatory disease, accelerating wound healing, **decreasing scarring** during wound healing, and **preventing** tumor angiogenesis and/or metastasis comprising administering to a subject in need of such **treatment** an effective amount of at least one peptide or compound based on a thrombin receptor sequence.

DRWD FIG. 6 illustrates the effects of **genistein** and indomethacin on the actions of P5-NH.sub.2 and carbachol in the RLM assay.

DETD . . . the healing process. The acceleration of would healing would also prove of value in the use of skin grafts for **treating** burn patients and in **treatment** of ocular injuries. It is likely that thrombin itself, generated at the site of injury, plays an important role in. . . healing process; alternatively, in some situations, one might wish to delay the healing process to avoid the formation of inappropriate **scarring**, leading to the formation of cheloid. Thus, for sites of injury and tissue repair, it could prove of use to. . . antagonists, depending on the situation. A non-degradable super-active analogue of the thrombin receptor peptide may be preferably used for such **treatment** in both humans and animals. The agents may also be used in the therapy of duodenal, ileal and colonic ulcer. . .

DETD . . . action of P5 in the GLM preparation was also selectively blocked by relatively low concentrations of the tyrosine kinase inhibitors, **genistein** (GS) and tyrphostin (TP)(14). We wished to determine if the active P5 analogues modified at the C-and N-terminus (represented by. . . E, F, G and H), the contractile actions of both P5--NH.sub.2 and Pr-P4 were blocked by either indomethacin or tyrphostin; **genistein** was similarly effective (not shown). Because of the desensitization caused by Pr-P4, in the experiments shown for the GLM in. . . tissue sensitivity to TRPs was evaluated by exposure to P5--NH.sub.2 (5 μg/mL; 7.8 μM) before determining the effects of tyrphostin, **genistein** and indomethacin on Pr-P4 action (G, H, FIG. 5). In contrast, the responses of the RA, which were abolished with. . . in FIG. 6, the response of the RLM preparation to P5-NH.sub.2 was also quite sensitive to tyrphostin (not shown) and **genistein** (77±3% inhibition: mean ±S.E.M for n=6) but was only partially affected by indomethacin (40±6% inhibition: mean ±S.E.M. for n=6). In. . .

DETD . . . indomethacin, noradrenaline and angiotensin-II and thrombin (human, 3000 to 4000 U/mg; 1 U/mL.perspectiveto.10 nM) were from Sigma (St. Louis, Mo.); **genistein** (GS) was from ICN Biochemicals, Costa Mesa, Calif. Tyrphostin (TP) (RG 50864, also designated AG213) was obtained through the courtesy of Dr. R. R. Swillo, Rhone-Poulenc Rorer (Collegeville, Pa). **Genistein** and tyrphostin were dissolved in dimethylsulfoxide (DMSO) to yield a stock solution that, when diluted, resulted in a concentration of. . .

PI US 5516889 19960514

ANSWER 51 OF 60 PCTFULL COPYRIGHT 2004 Univentio on STN

DETD Anti-acne actives can be effective in **treating** acne vulgaris, a chronic disorder of the pilosebaceous follicles. The condition involves inflammation of the pilosebaceous apparatus thereby resulting in lesions, which may include papules, pustules, cysts, comedomes, and severe **scarring**. The bacteria,Corynebacterium acnes and Staphylococcus epidermidis are usually present in the pustular contents.

. . .

from Laboratories Serobiologiques); formononetin; forsythia fruit extract; gallic acid esters; gamma butyric acid; GATULINE RC (available from Gattlefosse, located in Priest, France); **genistein**; genisteine; genistic acid; gingko bilboa extracts; ginseng extracts; ginsenoside (RO, R6-,, R6-2,R6-3, Rc, RD, RE] RP RF]29 P]6-1] RG-2); gluco pyranosyl-l-ascorbate; glutathione. . .

ACCESSION NUMBER: 1999055303 PCTFULL ED 20020515

TITLE (ENGLISH): CLEANSING ARTICLES FOR SKIN AND/OR HAIR WHICH ALSO DEPOSITS SKIN CARE ACTIVES

TITLE (FRENCH): PRODUITS D'HYGIENE POUR LA PEAU ET/OU LES CHEVEUX DEPOSANT DE PLUS DES INGREDIENTS ACTIFS TRAITANT LA PEAU

INVENTOR(S): ALBACARYS, Lourdes, Dessus;
McATEE, David, Michael;
DECKNER, George, Endel

PATENT ASSIGNEE(S): THE PROCTER & GAMBLE COMPANY;
ALBACARYS, Lourdes, Dessus;
McATEE, David, Michael;
DECKNER, George, Endel

LANGUAGE OF PUBL.: English

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9955303	A1	19991104

DESIGNATED STATES

W: AL AM AT AT AU AZ BA BB BG BR BY CA CH CN CU CZ CZ DE DE DK DK EE EE ES FI FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW GH GM KE LS MW SD SL SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 1999-IB635 A 19990412

PRIORITY INFO.: US 1998-60/083,015 19980424

L36 ANSWER 59 OF 149 USPATFULL on STN

ACCESSION NUMBER:	2001:229235 USPATFULL		
TITLE:	METHOD FOR USING SOLUBLE CURCUMIN TO INHIBIT PHOSPHORYLASE KINASE IN INFLAMMATORY DISEASES		
INVENTOR(S):	HENG, MADALENE C.Y., NORTHRIDGE, CA, United States		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001051184	A1	20011213
APPLICATION INFO.:	US 1999-315856	A1	19990520 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	ATTN: DAVID A. FARAH. M.D., SHELDON & MAK, 225 SOUTH LAKE AVENUE, SUITE 900, PASADENA, CA, 91101		
NUMBER OF CLAIMS:	115		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	13 Drawing Page(s)		
LINE COUNT:	4191		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

rs ,
inflammatory diseases and disorders, pyogenic granuloma, scleroderma, synovitis, trachoma and vascular adhesions.

DETD . . . and even antioxidants (U.S. Pat. No. 5,571,523; incorporated herein by reference) may also be used. Tyrosine kinase inhibitors, such as **genistein**, may also be linked to the agents of the present invention that target the cell surface receptor, VEGFR1 (as supported. . .

DETD The present invention may be used to **treat** animals and patients with aberrant angiogenesis, such as that contributing to a variety of diseases and disorders. The most prevalent and/or clinically important of these, outside the field of cancer **treatment**, include arthritis, rheumatoid arthritis, psoriasis, atherosclerosis, diabetic retinopathy, age-related macular degeneration, Grave's disease, vascular restenosis, including restenosis following angioplasty, arteriovenous. . . meningioma, hemangioma and neovascular glaucoma. Other potential targets for intervention include angiofibroma, atherosclerotic plaques, corneal graft neovascularization, hemophilic joints, hypertrophic **scars**, osler-weber syndrome, pyogenic granuloma retrolental fibroplasia, scleroderma, trachoma, vascular adhesions, synovitis, dermatitis, various other inflammatory diseases and disorders, and even endometriosis. Further diseases and disorders that are **treatable** by the invention, and the unifying basis of such angiogenic disorders, are set forth below.

ACCESSION NUMBER: 2002:19058 USPATFULL
TITLE: Antibody compositions for selectively inhibiting VEGF
INVENTOR(S): Thorpe, Philip E., Dallas, TX, United States
Brekken, Rolf A., Seattle, WA, United States
PATENT ASSIGNEE(S): Board of Regents, The University of Texas System, Austin, TX, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6342219	B1	20020129
APPLICATION INFO.:	US 2000-561500		20000428 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-131432P	19990428 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Chan, Christina Y.
ASSISTANT EXAMINER: Huynh, Phuong N.
LEGAL REPRESENTATIVE: Williams, Morgan and Amerson
NUMBER OF CLAIMS: 50
EXEMPLARY CLAIM: 20
NUMBER OF DRAWINGS: 7 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 10403
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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L36 ANSWER 55 OF 149 USPATFULL on STN

DETD . . . decrease the severity of symptoms; decrease the duration of disease exacerbations; increase the frequency and duration of disease remission/symptom-free periods; **prevent** fixed impairment and disability; and/or **prevent**/attenuate chronic progression of the disease. Clinically, this would result in improvement in visual symptoms (visual loss, diplopia), gait disorders (weakness,. . . dysfunction (weakness, spasticity, sensory loss), bladder dysfunction (urgency, incontinence, hesitancy, incomplete emptying), depression, emotional lability, and cognitive impairment. Pathologically the **treatment reduces** one or more of the following, such as myelin loss, breakdown of the blood-brain barrier, perivascular infiltration of mononuclear cells, immunologic abnormalities, gliotic **scar** formation and astrocyte proliferation, metalloproteinase production, and impaired conduction velocity.

DETD . . . Utilizing the agents, compositions and methods provided herein a wide variety of surgical adhesions and complications of surgery can be **treated** or **prevented**. Adhesion formation or unwanted **scar** tissue accumulation/encapsulation complicates a variety of surgical procedures. As described above, surgical adhesions complicate virtually any open or endoscopic surgical. . . the function of the surgical implant (e.g., breast implant, artificial joint, surgical mesh, vascular graft, dural patch). Chronic inflammation and **scarring** also occurs during surgery to correct chronic sinusitis or removal of other regions of chronic inflammation (e.g., foreign bodies, infections. . .

DETD . . . "film", or "wrap" which releases the drug over a period of time such that the incidence of surgical adhesions is **reduced**. During endoscopic procedures, the paclitaxel-polymer preparation is applied as a "spray", via delivery ports in the endoscope, to the mesentery. . . to 20% paclitaxel is applied to the surface of the surgical implant (e.g., breast implant, artificial joint, vascular graft) to **prevent** encapsulation/inappropriate **scarring** in the vicinity of the implant. In yet another preferred embodiment, a polymeric implant containing 0.1% to 20% paclitaxel by. . . cavity, chest cavity, abdominal cavity, or at the operative site during neurosurgery) such that recurrence of inflammation, adhesion formation, or **scarring** is **reduced**. In another embodiment, lavage fluid containing 1 to 75 mg/m.sup.2 (preferably 10 to 50 mg/m.sup.2) paclitaxel, would be used at. . .

DETD . . . paste (3 mg) 20% 0/10

MDHC (tyrosine inhibitor)	PCL paste (3 mg)	20%	0/8
erbstatin	PCL paste (3 mg)	20%	0/5 - too toxic
genistein	PCL paste (3 mg)	10%	0/7
	PCL paste (3 mg)	20%	0/4
herbimysin	PCL paste (3 mg)	2%	3/4
	PCL paste. . .		

DETD . . . polymer or a 2 mm thick disc of polymer, either of which may be applied to the tissue surface to **prevent** subsequent **scarring** and adhesion formation. This film was designed to be placed on exposed tissue so that any encapsulated drug can be. . .

ACCESSION NUMBER: 2002:22462 USPATFULL
TITLE: COMPOSITIONS AND METHODS FOR TREATING OR PREVENTING INFLAMMATORY DISEASES
INVENTOR(S): HUNTER, WILLIAM L., VANCOUVER, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002013298	A1	20020131
APPLICATION INFO.:	US 1999-368463	A1	19990804 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1998-88546, filed on 1 Jun

1998, PENDING Continuation-in-part of Ser. No. US 1997-980549, filed on 1 Dec 1997, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-32215P	19961202 (60)
	US 1997-63087P	19971024 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092	
NUMBER OF CLAIMS:	45	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	110 Drawing Page(s)	
LINE COUNT:	8318	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L36 ANSWER 47 OF 149 USPATFULL on STN

SUMM . . . harmless topical depigmenting substances with good efficacy. The use of such substances with good efficacy is most particularly desired for **treating** regional hyperpigmentations caused by melanocyte hyperactivity, such as idiopathic melasmas, occurring during pregnancy ("pregnancy mask" or chloasma) or during oestro-progestative. . . . post-lesional cicatrization, and also certain leukodermas, such as vitiligo. For these conditions (in which the cicatrizations can result in a **scar** which gives the skin a whiter appearance) and for leukodermas, failing the ability to repigment the damage to skin, the. . . .

DETD [0040] Isoflavonoids of natural origin are preferably used. Included are: daidzin, genistin, daidzein, formononetin, cuneatin, **genistein**, isoprunetin and prunetin, cajanin, orobol, pratensein, santal, junipegenin A, glycitein, afrormosin, retusin, tectorigenin, irisolidone, and jamaicin, and also analogues and. . . .

DETD [0041] Isoflavones are preferred for use in the present invention. This term covers both the aglycone forms (e.g., daidzein, **genistein** and glycitein) and the glycosylated forms (e.g., daidzine, genistine, glycitine) of the isoflavones.

CLM What is claimed is:

. . . composition according to claim 1, wherein said isoflavonoid is selected from the group consisting of daidzin, genistin, daidzein, formononetin, cuneatin, **genistein**, isoprunetin and prunetin, cajanin, orobol, pratensein, santal, junipegenin A, glycitein, afrormosin, retusin, tectorigenin, irisolidone, jamaicin, and mixtures thereof.

IT **446-72-0**, Genistein 480-23-9, Orobol 485-72-3, Formononetin 486-66-8, Daidzein 529-59-9, Genistin 529-60-2, Santal 548-77-6, Tectorigenin 550-79-8, Afrormosin 552-59-0, Prunetin 552-66-9, Daidzin 2284-31-3, Pratensein 2345-17-7, Irisolidone 4569-98-6, Isoprunetin 7159-95-7 7741-28-8, Cuneatin 21572-58-7 24211-36-7, Jamaicin 32884-36-9, Cajanin 37816-19-6, Retusin 40957-83-3, Glycitein 54734-40-6 76265-28-6, Junipegenin a 220717-78-2
(skin whitening cosmetic compns. comprising aminophenol derivative and isoflavonoid)

ACCESSION NUMBER: 2002:199172 USPATFULL

TITLE: Cosmetic composition containing an aminophenol derivative and an isoflavonoid

INVENTOR(S): Chevalier, Veronique, Villecresnes, FRANCE
Pham, Dang-Man, Sucy-En-Brie, FRANCE

PATENT ASSIGNEE(S): L'OREAL, Paris, FRANCE (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002107282	A1	20020808
APPLICATION INFO.:	US 2001-986885	A1	20011113 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	FR 2000-14479	20001110

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: OBLON SPIVAK MCCLELLAND MAIER & NEUSTADT PC, FOURTH FLOOR, 1755 JEFFERSON DAVIS HIGHWAY, ARLINGTON, VA, 22202

NUMBER OF CLAIMS: 20

EXEMPLARY CLAIM: 1

L36 ANSWER 41 OF 149 USPATFULL on STN

SUMM [0012] U.S. Pat. No. 6,228,887 to Kligman discloses **treating** such skin disorders as photodamage, hyperpigmentation, rosacea, and **scarring** topically with high strength retinoids at a concentration effective to cause desquamation. Retinoids activate the the epidermal growth factor receptor,. . . skin cells, which results in the desquamation sought by this patent. The clinical examples given in this patent only involve **treating** photodamaged skin.

DETD [0018] The art has addressed inflammation and **scarring** caused by acne as a secondary benefit to the **treatment** of the disease; that is, if the acne is cured the factors causing **scarring** will be eliminated. There is otherwise no **treatment** directed at **preventing scarring** from acne. Neither is there presently any direct **treatment** for the inflammation accompanying acne. The conventional **treatment** acts to **prevent** further problems by alleviating the cause of the acne; for example, a patient is **treated** with tetracycline, an antiobiotic, in hopes of killing the P. acnes, and the death of the bacteria will effectively end the inflammation and future **scarring**. Much as antipyretics, analgesics, decongestants, and antihistamines have been developed to **treat** the symptoms of colds and upper respiratory infections (as opposed to antibiotics and antivirals to kill off the invading bacteria and viruses), there is a need for **treatments** diminishing if not **preventing scarring** and inflammation in acne.

DETD . . . areas of injury, invade acne-affected skin, and release both a collagenase (MMP-8) and another protease (neutrophil elastase) that likely exacerbate **scarring**. Additionally, we have discovered that acne-affected skin has an elevated collagenase (MMP-1) level from resident skin cells that further exacerbates **scarring**. We have disclosed that **inhibition** of these dermal matrix-degrading enzymes with the use of MMP **inhibitors** (in addition to and including retinoids) can **lessen scarring** of acne-affected skin. Neutrophils circulate in the blood and therfore must be recruited by a signalling mechanism to induce their. . .

DETD [0029] Indirect MMP inhibitors include the kinase inhibitors **genistein** and quercetin (as described in U.S. Pat. No. 5,637,703, U.S. Pat. No. 5,665,367, and FR-A-2,671,724, and related compounds, as well. . .

DETD . . . inhibits proliferation, and induces apoptosis of acute myelogenous leukemia cells", Blood, vol. 94, no. 8, Oct. 15, 1999 (pp. 2844-53). **Genistein**: Tabary et al., "**Genistein** inhibits constitutive and inducible NFkappaB activation and decreases IL-8 production by human cystic fibrosis bronchial gland cells", Am J. Pathol,. . . 12-epi-scalaradial, and LY311727; arachidonyl methyl ketone analogue (AACOCH.sub.3) and the eicosapentanoyl analogue (EPACHOHCF.sub.3) had no effect on TNF-induced NF-κB activation. **Genistein**, erbstatin: Natarajan et aL, "Protein tyrosine kinase inhibitors block tumor necrosis factor-induced activation of nuclear factor-KB, degradation of IKBα, nuclear. . .

DETD [0033] **Inhibitors** of TLRs (toll-like receptors) and/or other receptors that are sensitive to the LPS-like compounds associated with **acne lesions** can be used to ameliorate the signalling that induces the cytokines TNFα, IL-1β, IL- 8, and IL-10, as shown in. . .

ACCESSION NUMBER: 2002:323230 USPATFULL
TITLE: Method and compositions for treating rosacea
INVENTOR(S): Kang, Sewon, Ann Arbor, MI, UNITED STATES
Voorhees, John J., Ann Arbor, MI, UNITED STATES
Fisher, Gary J., Ypsilanti, MI, UNITED STATES

NUMBER	KIND	DATE

PATENT INFORMATION: US 2002183399 A1 20021205
APPLICATION INFO.: US 2002-142724 A1 20020509 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-289758P	20010509 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Bradley N. Ruben, Suite 5A, 463 First Street, Hoboken, NJ, 07030
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
LINE COUNT: 896
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L31 ANSWER 58 OF 60 PCTFULL COPYRIGHT 2004 Univentio on STN

DETD . . . been shown to inhibit
apoptosis in cerebellar granule cells, although by distinct mechanisms
(Galli el al.,
1995). The protein tyrosine kinase inhibitors **genistein** and
herbimycin A have both
been shown to prevent anti-CD3 monoclonal antibody-induced thymic
apoptosis
(Migita et al., 1994). Interleukin-6 (IL-6) inhibits constitutive,. .
.
. . .
vivo. Inducing or
speeding up the overall regeneration process by administering
regenerated tissue to
the site of injury is beneficial as it **lessens** the chance that
the body will attempt to
repair the injury, leading to the formation of **scar** tissue and
the loss of normal tissue
structure and function.

ACCESSION NUMBER: 1997045533 PCTFULL ED 20020514
TITLE (ENGLISH): ENGINEERING ORAL TISSUES
TITLE (FRENCH): RECONSTITUTION DE TISSUS BUCCAUX
INVENTOR(S): MOONEY, David, J.;
RUTHERFORD, Robert, Bruce
PATENT ASSIGNEE(S): THE REGENTS OF THE UNIVERSITY OF MICHIGAN;
MOONEY, David, J.;
RUTHERFORD, Robert, Bruce
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

	NUMBER	KIND	DATE
	WO 9745533	A1	19971204

DESIGNATED STATES
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE
ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS
LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG
SI SK TJ TM TR TT UA UG US UZ VN YU GH KE LS MW SD SZ
UG AM AZ BY KG KZ MD RU TJ TM AT BE CH DE DK ES FI FR
GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN ML
MR NE SN TD TG

APPLICATION INFO.: WO 1997-US8977 A 19970528
PRIORITY INFO.: US 1996-60/018,450 19960528

L36 ANSWER 149 OF 149 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 96:891798 SCISEARCH
GA The Genuine Article (R) Number: VV058
TI Integrin alpha 2 beta 1-dependent contraction of floating collagen gels and induction of collagenase are inhibited by tyrosine kinase inhibitors
AU Broberg A; Heino J (Reprint)
CS UNIV TURKU, MEDICITY RES LAB, FIN-20520 TURKU, FINLAND (Reprint); UNIV TURKU, MEDICITY RES LAB, FIN-20520 TURKU, FINLAND; UNIV TURKU, DEPT BIOCHEM MED, FIN-20520 TURKU, FINLAND
CYA FINLAND
SO EXPERIMENTAL CELL RESEARCH, (10 OCT 1996) Vol. 228, No. 1, pp. 29-35. Publisher: ACADEMIC PRESS INC JNL-COMP SUBSCRIPTIONS, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0014-4827.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 39
AB A cell culture inside a three-dimensional gel of fibrillar collagen is an experimental model used to study the response of cells to the extracellular matrix. Many cell types induce the contraction of gel and simultaneously decrease their production of type I collagen, whereas the expression of interstitial collagenase (matrix metalloproteinase-1; MMP-1) is enhanced. me have previously shown that in osteogenic cells the collagen receptor alpha 2 beta 1 integrin is a positive regulator of MMP-1 and that the number of alpha 2 beta 1 integrins on the cell surface also regulates the magnitude of contraction. However, the downregulation of collagen mRNA levels is not initiated by alpha 2 beta 1 integrin. Here, we have studied in human KHOS-240 and MG-63 osteosarcoma cells and in human skin fibroblasts the effects of tyrosine kinase inhibitors on collagen gel contraction and on the regulation of MMP-1 and collagen alpha 1(I) genes by extracellular collagen. The induction of MMP-1 could be inhibited by all tyrosine kinase inhibitors tested with the exception of **genistein**. None of them could prevent the downregulation of collagen expression. Thus, the collagen-induced alterations in the expression of MMP-1 and collagen alpha 1(I) seem to be dependent on distinct signal transduction pathways. Many of the **inhibitors**, including **genistein**, could **prevent** the contraction of collagen gels. The effect was not related to their ability to **inhibit** cell growth, because an **inhibitor** specific for DNA synthesis and cell division did not have the same effect. Thus, we suggest that the process of collagen gel contraction requires protein-tyrosine phosphorylation and that the ability of cells to contract collagen gels is not related to the induction of MMP-1 or to the level of collagen alpha 1(I) expression. Finally, we propose that the tyrosine kinase **inhibitors** might be considered as candidate molecules in the **treatment** of pathological **scar** contraction. (C) 1996 Academic Press, Inc.
CC ONCOLOGY; CELL BIOLOGY
STP KeyWords Plus (R): GENE-EXPRESSION; SIGNAL-TRANSDUCTION; LATTICE CONTRACTION; FIBROBLASTS; RECEPTOR; BINDING; PROTEIN; SUBUNIT; DOMAIN; CELLS
RF 94-0081 002; NADPH OXIDASE ACTIVATION; SMALL GTP-BINDING PROTEINS; CHRONIC GRANULOMATOUS-DISEASE
94-0484 002; ADHESION MOLECULES; P-SELECTIN GLYCOPROTEIN LIGAND; LEUKOCYTE HOMING RECEPTORS
94-0971 001; MATRIX METALLOPROTEINASE EXPRESSION; TISSUE INHIBITOR; 92-KD TYPE-IV COLLAGENASE ACTIVITY; CULTURED VASCULAR SMOOTH-MUSCLE CELLS; PLASMINOGEN ACTIVATION
94-2201 001; COLLAGEN GEL CONTRACTION; FIBROBLASTS IN-VITRO; TRANSFORMING GROWTH-FACTOR-BETA
RE

Referenced Author	Year	VOL	PG	Referenced Work

(RAU)	(RPY)	(RVL)	(RPG)	(RWK)
BELKIN V M	1990	111	2159	J CELL BIOL
CHIRGWIN J M	1979	10	5291	BIOCHEMISTRY-US
CLARK E A	1995	268	233	SCIENCE
CLOVER J	1992	103	267	J CELL SCI
DANS M J	1994	102	118	J INVEST DERMATOL
EBLE J A	1993	12	4795	EMBO J
FORT P	1985	13	1431	NUCLEIC ACIDS RES
GARNER W L	1995	3	185	WOUND REP REGUL
GOLDBERG G I	1986	261	6600	J BIOL CHEM
GRINNELL F	1994	124	401	J CELL BIOL
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GULLBERG D	1990	186	264	EXP CELL RES
HEINO J	1996	65	717	INT J CANCER
HUHTALA P	1995	129	867	J CELL BIOL
HYNES R O	1992	69	11	CELL
KATAMA T	1994	269	9659	J BIOL CHEM
KERN A	1994	269	22811	J BIOL CHEM
KLEIN C E	1991	115	1427	J CELL BIOL
LANGHOLZ O	1995	131	1903	J CELL BIOL
LEE J O	1995	80	631	CELL
MAKELA J K	1988	16	349	NUCLEIC ACIDS RES TU
MIYAMOTO S	1995	267	883	SCIENCE
MONTESANO R	1988	85	4894	P NATL ACAD SCI USA
PROCKOP D J	1995	64	403	ANN REV BIOCH
RIDLEY A J	1992	70	389	CELL
RIIKONEN T	1995	270	376	J BIOL CHEM
RIIKONEN T	1995	270	13548	J BIOL CHEM
RUOSLAHTI E	1991	87	1	J CLIN INVEST
SAARIALHOKERE U K	1993	92	2858	J CLIN INVEST
SANTALA P	1994	269	1276	J BIOL CHEM
SCHIRO J A	1991	67	403	CELL
SELTZER J L	1994	213	365	EXP CELL RES
SUDBECK B D	1994	269	30022	J BIOL CHEM
TAKADA Y	1989	109	397	J CELL BIOL
TREMBLE P	1995	129	1707	J CELL BIOL
TURNER D C	1989	9	3287	J NEUROSCI
WERB Z	1989	109	877	J CELL BIOL
YAMATO M	1995	117	940	J BIOCHEM-TOKYO

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L36 ANSWER 58 OF 149 USPATFULL on STN

SUMM Anti-acne actives can be effective in **treating** acne vulgaris, a chronic disorder of the pilosebaceous follicles. The condition involves inflammation of the pilosebaceous apparatus thereby resulting in lesions, which may include papules, pustules, cysts, comedomes, and severe **scarring**. The bacteria Corynebacterium acnes and Staphylococcus epidermidis are usually present in the pustular contents.

SUMM Anti-wrinkle, anti-skin atrophy and skin repair actives can be effective in replenishing or rejuvenating the epidermal layer. These actives generally provide these desirable skin care benefits by promoting or maintaining the natural process of desquamation. Nonlimiting examples of antiwrinkle and anti-skin atrophy actives include retinoic acid and its derivatives (e.g., cis and trans); retinal; retinol; retinyl esters such as retinyl acetate, retinyl palmitate, and retinyl propionate; vitamin B.sub.3 compounds (such as niacinamide and nicotinic acid), salicylic acid and derivatives thereof (such as 5-octanoyl salicylic acid, heptyloxy 4 salicylic acid, and 4-methoxy salicylic acid); sulfur-containing D and L amino acids and their derivatives and salts, particularly the N-acetyl derivatives, a preferred example of which is N-acetyl-L-cysteine; thiols, e.g. ethane thiol; hydroxy acids, phytic acid, lipoic acid; lysophosphatidic acid; skin peel agents (e.g., phenol and the like); Actein 27-Deoxyactein Cimicifugoside (available from Cirnigoside); adapalene; ademethionine; adenosine; aletris extract; alkyl glutathione esters; alkoxyalkoxy alkoxyn benzoic and derivatives; aloe derived lectins; amino propane phosphoric acid; 3-aminopropyl dihydrogen phosphate; Amadorine (available from Barnet Products); anise extracts; AOSINE (available from Secma); arginine amino benzoate; ASC III (available from E. Merck, located in Darmstadt, Germany); ascorbic acid; ascorbyl palmitate; asiatic acid; asiaticosides; ARLAMOL GEO.TM. (available from ICI, located in Wilmington, Del.); azaleic acid; benzoic acid derivatives; bertholletia extracts; betulinic acid; BIOCHANIN A AND BIOPEPTIDE CL (available from Sederma, located in Brooklyn, N.Y.); BIOPEPTIDE EL (available from Sederma); biotin; blackberry bark extract; blackberry lily extracts; black cohosh extract; blue cohesh extract; butanoyl betulinic acid; carboxymethyl 1,3 beta glucan; catecholamnines; chalcones; citric acid esters; chaste tree extract; clover extracts; coumestrol; CPC Peptide (available from Barnet Products); daidzein; dang gui extract; darutoside; debromo laurinterol; 1-decanoyl-glycero-phosphonic acid; dehydrocholesterol; dehydrodicreosol; dehydrodieugenol; dehydroepiandersterone; DERMOLECTINE (available from Sederma); dehydroascorbic acid; dehydroepiandersterone sulfate; dianethole; dihydroxy benzoic acid; 2,4 dihydroxybenzoic acid; diglycol guanidine succinate; diosgenin; disodium ascorbyl phosphate; dodecanedioic acid; Ederline (available from Seporga); Enderline (available from Laboratories Seporga); equol; eriodictyol; estrogen and its derivatives; ETF (available from Laboratories Seporga); ethocyn; ELESERYL SH (available from Laboratories Serobiologiques, located in Somerville, N.J.); ENDONUCLEINE (available from Laboratories Serobiologiques); ergosterol; eythrobic acid; fennel extract; fenugreek seed extract; FIBRASTIL (available from Sederma); FIBROSTIMULINES S and P (available from Sederma); FIRMOGEN LS 8445 (available from Laboratories Serobiologiques); formononetin; forsythia fruit extract; gallic acid esters; gamma amino butyric acid; GATULINE RC (available from Gattlefosse, located in Priest, France); **genistein**; genisteine; genistic acid; gentisyl alcohol; gingko bilboa extracts; ginseng extracts; ginsenoside (RO, R.sub.6-1, R.sub.6-2, R.sub.6-3, R.sub.C, R.sub.D, R.sub.E, R.sub.F, R.sub.F-2, R.sub.G-1, R.sub.G-2); gluco pyranosyl-L-ascorbate; glutathione and its esters; glycitein; hesperitin; hexahydro curcumin; HMG- coenzyme A reductase inhibitors; hops extracts; 11 hydroxy undecanoic acid; 10 hydroxy decanoic acid; 25-hydroxycholesterol; 7-hydroxylated sterols; hydroxyethyl isostearyloxy isopropanolamine; hydroxy-tetra methyl piperidinyloxy;

hypotaurine; ibukijakou extract; isoflavone SG 10 (available from Barnet Products); kinetin; kohki extract; L-2-OXO-thiazolidine-4-carboxylic acid esters; lactate dehydrogenase inhibitors; 1-lauryl, -lyso-phosphatidyl choline; lectins; lichochalcone LF15 (available from Maruzen); licorice extracts; lignan; lumisterol; lupenes; luteolin; lysophosphitidic acid; magnesium ascorbyl phosphate; margin; melatonin; melibiose; metalloproteinase inhibitors; methoprene; methoprenic acid; mevalonic acid; MPC COMPLEX (available from CLR); N methyl serine; N methyl taurine; N, N.sup.1-bis (lactyl) cysteamine; naringenin; neotigogenin; o-desmethylangoiensin; oat beta glucan; oleanolic acid; pantethine; phenylalanine; photoanethone; piperdine; placental extracts; pratensein; pregnenolone; pregnenolone acetate; pregnenolone succinate; premarin; quillaic acid; raloxifene; REPAIR FACTOR 1 and REPAIR FACTOR FCP (both available from Sederna); retinoates (esters of C.sub.2-C.sub.20 alcohols); retinyl glucuronate; retinyl linoleate; S-carboxymethyl cysteine; SEANAMINE FP (available from Laboratories Serobiologiques); sodium ascorbyl phosphate; soya extracts; spleen extracts; tachysterol; taurine; tazarotene; tempol; thymulen; thymus extracts; thyroid hormones; tigogenin; tocopheryl retinoate; toxifolin; traumatic acid; tricholine citrate; trifoside; uracil derivatives; ursolic acid; vitamin D.sub.3 and its analogs; vitamin K; vitex extract; yam extract; yamogenin; zeatin; and mixtures thereof.

ACCESSION NUMBER: 2002:9654 USPATFULL

TITLE: Cleansing articles for skin and/or hair which also deposit skin care actives

INVENTOR(S): Albacarys, Lourdes Dessus, West Chester, OH, United States
McAtee, David Michael, Mason, OH, United States
Deckner, George Endel, Cincinnati, OH, United States
PATENT ASSIGNEE(S): The Procter & Gamble Company, Cincinnati, OH, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6338855	B1	20020115
APPLICATION INFO.:	US 1999-296334		19990422 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1998-65991, filed on 24 Apr 1998, now abandoned Continuation-in-part of Ser. No. US 1997-974033, filed on 19 Nov 1997, now abandoned Continuation-in-part of Ser. No. US 1996-738145, filed on 25 Oct 1996, now abandoned Continuation of Ser. No. US 1996-738668, filed on 25 Oct 1996, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-83015P	19980424 (60)

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PRIMARY EXAMINER: Criares, Theodore J.
LEGAL REPRESENTATIVE: Allen, George W., Matthews, Armina E., Tsuneki, Fumiko
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 0 Drawing Figure(s); 0 Drawing Page(s)
LINE COUNT: 3405
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L36 ANSWER 68 OF 149 USPATFULL on STN

DETD . . . to inhibit apoptosis in cerebellar granule cells, although by distinct mechanisms (Galli et al., 1995). The protein tyrosine kinase inhibitors **genistein** and herbimycin A have both been shown to prevent anti-CD3 monoclonal antibody-induced thymic apoptosis (Migita et al., 1994). Interleukin-6 (IL-6). . .

DETD . . . or speeding up the overall regeneration process by administering regenerated tissue to the site of injury is beneficial as it **lessens** the chance that the body will attempt to repair the injury, leading to the formation of **scar** tissue and the loss of normal tissue structure and function.

PI US 5885829 19990323

LL on STN

DETD . . . inflammatory diseases including but not limited to rheumatoid arthritis, inflammatory bowel disease, multiple sclerosis, psoriasis and asthma; wound healing and **scar reduction**; and neurodegenerative diseases. Therefore, the development of cell-based assays that provide information on the effect of a test compound on. . .

DETD . . . of tumor growth and metastasis, angiogenesis, thrombosis, restenosis, vascular overgrowth during macular degeneration, foam cell formation, inflammatory diseases, wound healing, **scar reduction**, and neurodegenerative diseases.

DETD . . . 10 μM Calphostin C (a protein kinase C inhibitor), 10 μM KN-52 (a Calmodulin dependent protein kinase inhibitor), 10 μM **genistein** (a tyrosine kinase inhibitor) and 10 nM wortmannin (a phosphatidyl inositol kinase inhibitor. These agents were also applied during measurement. . . different from the profile for cell motility. Calphostin C, for example, was equally effective at inhibiting cell spreading and movement, **genistein** and KN-62 were ineffective at the concentrations used at blocking either cellular response, and Wortmannin was more effectve at blocking. . .

CLM What is claimed is:

. . . of tumor growth and metastasis, angiogenesis, thrombosis, restenosis, vascular overgrowth during macular degeneration, foam cell formation, inflammatory diseases, wound healing, **scar reduction** , and neurodegenerative diseases.

. . . of tumor growth and metastasis, angiogenesis, thrombosis, restenosis, vascular overgrowth during macular degeneration, foam cell formation, inflammatory diseases, wound healing, **scar reduction** , and neurodegenerative diseases.

. . . of tumor growth and metastasis, angiogenesis, thrombosis, restenosis, vascular overgrowth during macular degeneration, foam cell formation, inflammatory diseases, wound healing, **scar reduction** , and neurodegenerative diseases.

PI US 2001041347 A1 20011115|
US 6716588 B2 20040406|

LL on STN

DETD . . . inflammatory diseases including but not limited to rheumatoid arthritis, inflammatory bowel disease, multiple sclerosis, psoriasis and asthma; wound healing and **scar reduction**; and neurodegenerative diseases. Therefore, the development of cell-based assays that provide information on the effect of a test compound on. . .

DETD . . . of tumor growth and metastasis, angiogenesis, thrombosis, restenosis, vascular overgrowth during macular degeneration, foam cell formation, inflammatory diseases, wound healing, **scar reduction**, and neurodegenerative diseases.

DETD . . . 10 μM Calphostin C (a protein kinase C inhibitor), 10 μM KN-52 (a Calmodulin dependent protein kinase inhibitor), 10 μM **genistein** (a tyrosine kinase inhibitor) and 10 nM wortmannin (a phosphatidyl inositol kinase inhibitor. These agents were also applied during measurement. . . different from the profile for cell motility. Calphostin C, for example, was equally effective at inhibiting cell spreading and movement, **genistein** and KN-62 were ineffective at the concentrations used at blocking either cellular response, and Wortmannin was more effectve at blocking. . .

CLM What is claimed is:

. . . of tumor growth and metastasis, angiogenesis, thrombosis, restenosis, vascular overgrowth during macular degeneration, foam cell formation, inflammatory diseases, wound healing, **scar reduction** , and neurodegenerative diseases.

. . . of tumor growth and metastasis, angiogenesis, thrombosis, restenosis, vascular overgrowth during macular degeneration, foam cell formation, inflammatory diseases, wound healing, **scar reduction** , and neurodegenerative diseases.

. . . of tumor growth and metastasis, angiogenesis, thrombosis, restenosis, vascular overgrowth during macular degeneration, foam cell formation, inflammatory diseases, wound healing, **scar reduction** , and neurodegenerative diseases.

PI US 2001041347 A1 20011115
US 6716588 B2 20040406

L28 ANSWER 12 OF 14 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 96321992 EMBASE
DN 1996321992
TI Integrin α2β1-dependent contraction of floating collagen gels and induction of collagenase are inhibited by tyrosine kinase inhibitors.
AU Broberg A.; Heino J.
CS Department of Medical Biochemistry, MediCity Research Laboratory, University of Turku, Tykistokatu 6A,FIN-20520 Turku, Finland
SO Experimental Cell Research, (1996) 228/1 (29-35).
ISSN: 0014-4827 CODEN: ECREAL
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
AB A cell culture inside a three-dimensional gel of fibrillar collagen is an experimental model used to study the response of cells to the extracellular matrix. Many cell types induce the contraction of gel and simultaneously decrease their production of type I collagen, whereas the expression of interstitial collagenase (matrix metalloproteinase-1; MMP-1) is enhanced. We have previously shown that in osteogenic cells the collagen receptor α2β1 integrin is a positive regulator of MMP-1 and that the number of α2β1 integrins on the cell surface also regulates the magnitude of contraction. However, the downregulation of collagen mRNA levels is not initiated by α2β1 integrin. Here, we have studied in human KHOS-240 and MG- 63 osteosarcoma cells and in human skin fibroblasts the effects of tyrosine kinase inhibitors on collagen gel contraction and on the regulation of MMP-1 and collagen α1(I) genes by extracellular collagen. The induction of MMP-1 could be inhibited by all tyrosine kinase inhibitors tested with the exception of **genistein**. None of them could prevent the downregulation of collagen expression. Thus, the collagen-induced alterations in the expression of MMP-1 and collagen α1(I) seem to be dependent on distinct signal transduction pathways. Many of the inhibitors, including **genistein**, could prevent the contraction of collagen gels. The effect was not related to their ability to inhibit cell growth, because an inhibitor specific for DNA synthesis and cell division did not have the same effect. Thus, we suggest that the process of collagen gel contraction requires protein-tyrosine phosphorylation and that the ability of cells to contract collagen gels is not related to the induction of MMP-1 or to the level of collagen α1(I) expression. Finally, we propose that the tyrosine kinase inhibitors might be considered as candidate molecules in the treatment of pathological **scar** contraction.
CT Medical Descriptors:
*extracellular matrix
article
collagen synthesis
controlled study
enzyme induction
enzyme phosphorylation
human
human cell
priority journal
protein expression
Drug Descriptors:
*collagen gel
*collagenase: EC, endogenous compound
*integrin
*protein tyrosine kinase inhibitor
collagen type 1
RN (collagenase) 9001-12-1

L7 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1996:746408 CAPLUS
DN 126:14769
ED Entered STN: 20 Dec 1996
TI Pharmaceutical composition containing substance inhibiting HSP47 production
IN Kiyosuke, Yoichi; Shirakami, Toshiharu; Morino, Masayoshi; Yoshikumi, Chikao
PA Kureha Chemical Industry Co., Ltd., Japan
SO Eur. Pat. Appl., 23 pp.
CODEN: EPXXDW
DT Patent
LA English
IC ICM A61K035-78
ICS A61K031-35; A61K031-70; A61K035-84; A61K038-00; A61K031-355; A61K031-19
CC 1-10 (Pharmacology)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 742012	A2	19961113	EP 1996-107224	19960508
	EP 742012	A3	19990908		
	R: DE, FR, GB, NL				
	JP 08301757	A2	19961119	JP 1995-136028	19950510
	JP 08301784	A2	19961119	JP 1995-136029	19950510
	JP 2892300	B2	19990517		
	JP 09012459	A2	19970114	JP 1995-186302	19950629
	JP 3003978	B2	20000131		
	JP 09040556	A2	19970210	JP 1995-210935	19950727
	JP 09040553	A2	19970210	JP 1995-211274	19950728
	JP 2933511	B2	19990816		
	CA 2175985	AA	19961111	CA 1996-2175985	19960507
	AU 9652140	A1	19961219	AU 1996-52140	19960507
	AU 689036	B2	19980319		
PRAI	JP 1995-136027	A	19950510		
	JP 1995-136028	A	19950510		
	JP 1995-136029	A	19950510		
	JP 1995-186302	A	19950629		
	JP 1995-210935	A	19950727		
	JP 1995-211274	A	19950728		

AB A pharmaceutical composition for treatment of liver cirrhosis, interstitial lung disease, chronic renal failure, **scars**, scleroderma, arteriosclerosis, and rheumatoid arthritis contains a substance which inhibits HSP47 production, selected from a malt extract, a flavonoid compound, a protein-bound polysaccharide from Coriolus versicolor, a paeoniflorin derivative, a tocopherol derivative, and a ferulic acid derivative HSP47 is apparently involved in processing of procollagen to collagen. The composition can efficiently improve the physiol. condition of a patient exhibiting overprodn. of the extracellular matrix, and is useful for preventing or treating various diseases accompanied with abnormal growth of the vascularization. Thus, HSP47 production by human uterus cancer cell line HeLa S3 was inhibited in vitro by paeoniflorin (100 mM), α-tocopherol (20 μM), and ferulic acid (100 μM).
ST collagen overprodn HSP47 protein inhibitor; extracellular matrix prodn HSP47 protein
IT Neoplasm
(HSP47 formation by cells of; substances inhibiting HSP47 production for treatment of extracellular matrix overprodn.)
IT Glycoproteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(PS-K; substances inhibiting HSP47 production for treatment of

extracellular matrix overprodn.)

IT Glycoproteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(collagen-binding, hsp 47; substances inhibiting HSP47 production for treatment of extracellular matrix overprodn.)

IT Rice (Oryza sativa)
(extract; substances inhibiting HSP47 production for treatment of extracellular matrix overprodn.)

IT Tea products
(exts.; substances inhibiting HSP47 production for treatment of extracellular matrix overprodn.)

IT Proteoglycans, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(of Coriolus versicolor; substances inhibiting HSP47 production for treatment of extracellular matrix overprodn.)

IT Peony (Paeonia)
(root extract; substances inhibiting HSP47 production for treatment of extracellular matrix overprodn.)

IT Malt
Trametes versicolor
(substances inhibiting HSP47 production for treatment of extracellular matrix overprodn.)

IT Flavanols
Flavonoids
Tocopherols
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(substances inhibiting HSP47 production for treatment of extracellular matrix overprodn.)

IT Antibodies
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(to heat shock protein HSP47; substances inhibiting HSP47 production for treatment of extracellular matrix overprodn.)

IT 59-02-9, α-Tocopherol **117-39-5**, Quercetin 153-18-4, Rutin 490-46-0, Epicatechin 491-67-8, Baicalein 863-03-6, Epicatechin gallate 970-74-1, Epigallocatechin 989-51-5, Epigallocatechin gallate 1135-24-6, Ferulic acid 1135-24-6D, Ferulic acid, derivs. 23180-57-6, Paeoniflorin 23180-57-6D, Paeoniflorin, derivs.
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); **BIOL (Biological study)**; **USES (Uses)**
(substances inhibiting HSP47 production for treatment of extracellular matrix overprodn.)